

action of the intragranular electron transport system with the externally reduced coenzymes, as an hypothesis one may assume the involvement of some structural factor or/and of a carrier system (diaphorase, quinone) scarcely participating in the respiration of resting cells.

Riassunto. Gli autori dimostrano che l'aumento della respirazione dei leucociti durante la fagocitosi non è dovuto a fuoriuscita di una NADH-ossidasi dai granuli, ma

alla maggiore attività NADH e soprattutto NADPH-ossidasi localizzata in granuli non lisati.

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The Effect of ACTH on Cholinesterase Activity in Plasma, Whole Blood, and Blood Cells of Rats

In 1960 one of us proved that when ACTH was administered to normal subjects and asthmatic patients, it produced increased cholinesterase activity in plasma, whole blood, and blood cells¹.

For this present study we used Wistar rats weighing about 250 g each. Blood was extracted by puncturing the jugular vein on four successive occasions: (a) under basic conditions, (b) and (c) after intramuscular and intraperitoneal injection of 10 units of ACTH, (d) under basic conditions. In this way the rats acted as their own controls.

Cholinesterase activity was determined in plasma, whole blood and blood cells by BIGG's colorimetric method².

ACTH was administered in the following way: 6 gel units intramuscularly and 4 units in a saline solution intraperitoneally.

Blood was extracted between 20 min and 120 min after the intraperitoneal injection of ACTH and from 3 to 5 h after its injection by the intramuscular route. This technique produced maximum stimulating effect of the cor-

ticotrophin on cholinesterase activity. Intramuscular and intraperitoneal injections were also given separately, but with this method we obtained less variation.

Extractions were performed weekly and blood tests performed in order to avoid anaemia in the experimental animals.

The averages obtained under basic conditions, and expressed in units of cholinesterase activity were: 92 ± 3.7 , 148 ± 3.9 , and 199 ± 4.3 in plasma, whole blood, and blood cells respectively (Figure).

The values rose to 97, 171, and 252 after the first administration of ACTH. After the second administration, using the same technique, cholinesterase activity again increased, to 118, 185, and 253. The general average under the action of ACTH was 108 ± 4.5 , 178 ± 2.4 , and 255 ± 3.3 in plasma, whole blood, and blood cells respectively. Analysis of the statistics showed a highly significant increase, $P: 0.001$, in the different blood fractions.

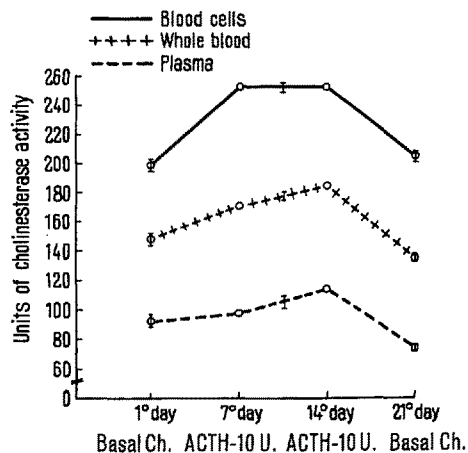
Cholinesterase activity was again determined 7 days after the last administration of ACTH and the values were found to have dropped to the basic figures: 72, 135, and 205 units of cholinesterase activity in plasma, whole blood, and cells, respectively.

According to these findings ACTH possesses a stimulating effect on cholinesterase activity both in man and rats.

Résumé. On a déterminé chez des rats l'activité cholinestérasiqne dans le plasma, sang total et cellules sanguines avant et après l'administration d'ACTH. On a constaté une augmentation hautement significative: $P: 0,001$ après l'administration de cette hormone.

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Cholinesterase activity in basal conditions and after ACTH.

Stimulation of Dorsal Axial Differentiation in Ventral Explants of *Rana pipiens* Embryos by CaCl_2 and Guanidine HCl

It has been demonstrated that exposure of ectoderm of the early salamander gastrula *in vitro* to saline solutions of pH 4.0 and 9.7 for brief periods can stimulate the differentiation of parts of the brain¹, while brief treatment of ventral mesoderm at pH 12.0 promotes the formation

of notochord, muscle and pronephric tubules². The means by which the extreme pH levels cause prospective ventral tissue to form dorsal axial structures possibly may be allied with the provision of soluble protein, and possibly some ribonucleic acid, from some of the yolk platelets

¹ J. R. VACCAREZZA and L. PELTZ, Presse Méd. 68, 723 (1960).

² H. G. BIGGS, S. CAREY, and D. B. MORRISON, Amer. J. clin. Path. 30, 181 (1958).

³ T. YAMADA, Biol. Bull. 98, 98 (1950).